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Risk for Sudden Cardiac Death Associated With Marathon Running

As an avid runner and occasional marathoner, I was pleased to learn that the risk of sudden death during or immediately after running a marathon is very low (1). Unfortunately, I believe that the authors' assessment of risk relative to other activities is somewhat misleading.

Assuming that there are 200,000,000 U.S. adults at risk for sudden death, and that there are 500,000 sudden deaths annually, the risk of sudden death or cardiac arrest as a function of living hours is approximately 1 death/3,504,000 h. The rate reported by Maron et al. (1) during or after a marathon, 1 death/215,000 h, is roughly 16-fold higher than the rate during normal living.

Thus, although the risk of dying suddenly during a marathon is quite low, it is still 16 times greater than the risk of sudden death during all other living activities combined.

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Reply

We thank Rich for his comments. We certainly agree, based on our recently published data (1), that the risk for sudden death directly associated with long-distance or marathon running is indeed exceedingly low (i.e., ~1 in 50,000). However, calculation of the overall risk for premature death associated with living can prove to be a rather complex and difficult undertaking. In our report (1) we chose to calculate such values directly from U.S. Vital Statistics and the National Center for Health Statistics for the years 1979, 1990 and 1991 (2). Perhaps this methodology accounts for the differences between our published values (1) and the estimates offered by Rich in his letter.

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Late Potentials in the Thrombolytic Era: Time for Reevaluation?

We read with interest the report by Karam et al. (1). The authors reported a decrease in the prevalence of late potentials with mechanical reperfusion compared with thrombolysis after myocardial infarction. However, the report raises a number of important issues.

Although the significance of late potentials in the early phase (<48 h) is unclear, El-Sherif et al. (2) found that only late potentials recorded between days 6 and 30 were associated with arrhythmic events. In particular, they recommended that "the optimal time window for obtaining a signal-averaged ECG is between 6 and 14 days after infarction." However, the study by El-Sherif et al. predated the thrombolytic era and excluded patients >79 years old. Although Karam et al. justify their recording time on this study, they give only the median time and neglect to give the range. A median recording time of 11 days suggests that many recordings may have been made after day 14, at which time late potentials have little prognostic significance. Furthermore, the authors fail to give any data on arrhythmic events or sudden death in the patients studied.

This leads to a more fundamental issue, which is the unproved role of late potentials as a predictor of arrhythmic events in a thrombolyzed cohort. Many of the pioneering studies referenced in the report predate the thrombolytic era. The widespread use of thrombolysis and early revascularization have significantly reduced the arrhythmic event and mortality rates after acute myocardial infarction (3,4). These same studies (neither of which is referenced) have failed to show a relation between late potential development and arrhythmic events in a thrombolyzed patient cohort.

We previously showed (5) that late potentials in the first 7 days after acute anterior myocardial infarction (after thrombolysis) are associated with an increase in ventricular volume at 6 weeks. In a larger study of patients receiving thrombolyic therapy, Hohnloser et al. (4) also found that late potentials were associated with wall motion abnormalities. On the basis of our findings, we suggested that the prognostic value of late potentials in the first week (and particularly on day 3) may be related to ventricular dilation. Although Karam et al. performed radionuclide left ventricular angiography in all patients, no